Albendazole-induced Recurrent Hepatitis

A 5-year-old male child presented with repeated episodes of acute-hepatitis, each episode occurring after 2-3 days of administering albendazole. He presented to us during the fourth such episode with complaints of acute onset fever, anorexia and vomiting followed by yellowish discoloration of eyes and urine. Each episode lasted 2-3 weeks, the intervening periods remaining uneventful. Liver was palpable 3.5 cm below the right costal margin. It was mildly tender and soft. There were no signs of chronic liver disease. Serum bilirubin on admission was 11.5 mg/dL (Direct- 9.5 and Indirect-2.0). Serum alanine transaminase, aspartate transaminase, alkaline phosphatase and gamma glutamyl transpeptidase (GGT) were 2720 IU/L, 4100 IU/L, 1247 IU/L and 26 IU/L, respectively. Albumin and globulin levels were 3.3 g/dL and 3.0 g/dL, respectively. Prothrombin time was 22 seconds (INR-1.6, control 12.6 secs) and aPTT was 31.0 secs (N-25-35 sec). Ceruloplasmin level was 35.64 mg/dL (N >20 mg/dL). HBsAg, anti HCV Ab, IgM HAV and IgM HEV, antinuclear antibodies, anti LKM antibody and anti smooth muscle antibody were negative. His condition improved within 2 weeks with subsidence of jaundice and hepatomegaly. On follow up, at 2 months, he was asymptomatic without hepatomegaly and with normal levels of bilirubin.

Albendazole (methyl 5-propylthio-2-benzimidazole-carbamate) is a widely used broad spectrum antihelminthic drug. Mild adverse effects like nausea, vomiting and pruritus have been occasionally reported [1]. However, reports of albendazole induced significant liver toxicity are rare. Moreover, most of the previous incidences have been reported following prolonged administration [1]. Recurrent hepatitis following single dose administration of albendazole is rare [2].

As all the common etiological markers of chronic and recurrent hepatitis were negative and due to temporal relation of albendazole ingestion with onset of self-limiting clinical jaundice four times in two years, a possibility of albendazole induced idiosyncratic hepatotoxicity was considered. He scored 5 on Naranjo Scale [3], categorizing as probable ADR. On Roussel Uclaf Causality Assessment Method of the Council for International Organizations of Medical Sciences scale [4,5], this child scored 9 points categorizing as ‘highly probable’ association of albendazole with DILI (drug induced liver injury).

We, as clinicians, need to be aware of this rare but significant adverse effect of this commonly and often empirically used drug.

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REFERENCES